

A. Harris  
627694

=> fil reg

COST IN U.S. DOLLARS

SINCE FILE

TOTAL

ENTRY

SESSION

FULL ESTIMATED COST

0.60

0.60

FILE 'REGISTRY' ENTERED AT 15:08:15 ON 23 APR 2001

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STRUCTURE FILE UPDATES: 22 APR 2001 HIGHEST RN 332014-61-6

DICTIONARY FILE UPDATES: 22 APR 2001 HIGHEST RN 332014-61-6

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Please note that search-term pricing does apply when  
conducting SmartSELECT searches.

Structure search limits have been increased. See HELP SLIMIT  
for details.

=> e gp75/cn 5

E1 1 GP63 SURFACE PROTEASE (LEISHMANIA INFANTUM)/CN  
E2 1 GP64 GLYCOPROTEIN (POLYSPHONDYLIUM PALLIDUM CLONE C-P644  
PRE

CURSOR)/CN

E3 0 --> GP75/CN

E4 2 GPA/CN

E5 1 GPA (AMIDE)/CN

=> fil medl,caplus,biosis,embase,wpids,jicst,scisearch

COST IN U.S. DOLLARS

SINCE FILE

TOTAL

ENTRY

SESSION

FULL ESTIMATED COST

0.31

0.91

FILE 'MEDLINE' ENTERED AT 15:08:37 ON 23 APR 2001

FILE 'CAPLUS' ENTERED AT 15:08:37 ON 23 APR 2001

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FILE 'EMBASE' ENTERED AT 15:08:37 ON 23 APR 2001

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FILE 'WPIDS' ENTERED AT 15:08:37 ON 23 APR 2001

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=> s (non human or insect)(2a)cell line and (human differ? antigen or human melanocyte? or gp75 or gp 75)

L1 0 FILE MEDLINE  
L2 0 FILE CAPLUS  
L3 0 FILE BIOSIS  
L4 0 FILE EMBASE  
L5 1 FILE WPIDS  
L6 0 FILE JICST-EPLUS  
L7 0 FILE SCISEARCH

TOTAL FOR ALL FILES

L8 1 (NON HUMAN OR INSECT)(2A) CELL LINE AND (HUMAN DIFFER? ANTIGEN OR HUMAN MELANOCYTE? OR GP75 OR GP 75)

=> d

L8 ANSWER 1 OF 1 WPIDS COPYRIGHT 2001 DERWENT INFORMATION LTD  
AN 1998-348236 [30] WPIDS  
DNC C1998-107615  
TI Stimulating immune response to differentiation antigen - which is expressed by tumour, comprises use of altered differentiation antigen expressed in, or derived from, another species.  
DC B04 D16  
IN HOUGHTON, A; NAFTZGER, C; VIJAYASARADHI, S  
PA (SLOK) SLOAN KETTERING INST CANCER RES  
CYC 21  
PI WO 9825574 A2 19980618 (199830)\* EN 19p A61K000-00  
RW: AT BE CH DE DK ES FI FR GB GR IE IT LU MC NL PT SE  
W: CA JP US  
EP 959893 A2 19991201 (200001) EN A61K035-12  
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU NL PT SE  
ADT WO 9825574 A2 WO 1997-US22669 19971210; EP 959893 A2 EP 1997-954555 19971210, WO 1997-US22669 19971210  
FDT EP 959893 A2 Based on WO 9825574  
PRAI US 1997-36419 19970217; US 1996-32535 19961210  
IC ICM A61K000-00; A61K035-12  
ICS A61K035-56; C12N005-06; C12N005-10; C12N005-18; C12N005-22; C12N015-06; C12N015-12; C12N015-63; C12N015-85

=> s immun? respons? and different? antigen? and antigen?

L9 313 FILE MEDLINE  
L10 231 FILE CAPLUS  
L11 348 FILE BIOSIS  
L12 376 FILE EMBASE  
L13 24 FILE WPIDS  
L14 237 FILE JICST-EPLUS  
L15 294 FILE SCISEARCH

TOTAL FOR ALL FILES

L16 1823 IMMUN? RESPONS? AND DIFFERENT? ANTIGEN? AND ANTIGEN?

=> s (gp75 or gp 75) and l16

L17 4 FILE MEDLINE  
 L18 6 FILE CAPLUS  
 L19 6 FILE BIOSIS  
 L20 5 FILE EMBASE  
 L21 0 FILE WPIDS  
 L22 0 FILE JICST-EPLUS  
 L23 5 FILE SCISEARCH

TOTAL FOR ALL FILES

L24 26 (GP75 OR GP 75) AND L16

=> s l24 not 18

L25 4 FILE MEDLINE  
 L26 6 FILE CAPLUS  
 L27 6 FILE BIOSIS  
 L28 5 FILE EMBASE  
 L29 0 FILE WPIDS  
 L30 0 FILE JICST-EPLUS  
 L31 5 FILE SCISEARCH

TOTAL FOR ALL FILES

L32 26 L24 NOT L8

=> dup rem l32

PROCESSING COMPLETED FOR L32

L33 7 DUP REM L32 (19 DUPLICATES REMOVED)

=> d 1-7 cbib abs;s houghton a?/au,in;s naftger c?/au,in;s sataluri  
 v?/au,in;s gregor p?/au,in

L33 ANSWER 1 OF 7 CAPLUS COPYRIGHT 2001 ACS

1998:402278 Document No. 129:66831 Method and compositions for stimulation  
 of an **immune response** to a **differentiation**  
**antigen** stimulated by an altered **differentiation**  
**antigen**. Houghton, Alan; Naftzger, Clarissa; Vijayasaradhi,  
 Setaluri (Sloan-Kettering Institute for Cancer Research, USA; Houghton,  
 Alan; Naftzger, Clarissa; Vijayasaradhi, Setaluri). PCT Int. Appl. WO  
 9825574 A2 19980618, 19 pp. DESIGNATED STATES: W: CA, JP, US; RW: AT,  
 BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE.  
 (English). CODEN: PIXXD2. APPLICATION: WO 1997-US22669 19971210.  
 PRIORITY: US 1996-32535 19961210; US 1997-36419 19970217.

AB Tolerance of the immune system for self-**differentiation**  
**antigens** can be overcome and an **immune response**  
 stimulated by administration of a therapeutic **differentiation**  
**antigen**. The therapeutic **differentiation**  
**antigen** is altered with respect to the target  
**differentiation antigen** in the individual being treated  
 (i.e., the **differentiation antigen** to which an  
**immune response** is desired) in one of three ways.  
 First, the therapeutic **differentiation antigen** may be  
 syngeneic with the target **differentiation antigen**,  
 provided that therapeutic **differentiation antigen** is  
 expressed in cells of a species different from the individual being  
 treated. For example, a human **differentiation antigen**  
 expressed in insect or other non-human host cells can be used to  
 stimulate

an immune response to the differentiation antigen in a human subject. Second, the therapeutic differentiation antigen may be a mutant form of a syngeneic differentiation antigen, for example a glycosylation mutant. Third, the therapeutic differentiation antigen may be a differentiation antigen (wild-type or mutant) of the same type from a species different from the individual being treated. For example, a mouse differentiation antigen can be used to stimulate an immune response to the corresponding differentiation antigen in a human subject. Administration of altered antigens in accordance with the invention results in an effective immunity against the original antigen expressed by the cancer in the treated individual.

- L33 ANSWER 2 OF 7 MEDLINE DUPLICATE 1  
 1999031212 Document Number: 99031212. PubMed ID: 9813669.  
 Adenovirus-mediated expression of melanoma antigen gp75  
 as immunotherapy for metastatic melanoma. Hirschowitz E A; Leonard S;  
 Song W; Ferris B; Leopold P L; Lewis J J; Bowne W B; Wang S; Houghton A N;  
 Crystal R G. (Division of Pulmonary and Critical Care Medicine, New York  
 Hospital-Cornell Medical Center, NY 10021, USA. ) GENE THERAPY, (1998  
 Jul) 5 (7) 975-83. Journal code: CCE; 9421525. ISSN: 0969-7128. Pub. country:  
 ENGLAND: United Kingdom. Language: English.  
 AB Melanocyte differentiation antigens, such as the brown  
 locus protein gp75, are potential biological targets for  
 immunotherapy. We investigated whether expression of the murine  
 gp75 cDNA mediated by an adenovirus (Ad) vector could induce  
 melanoma rejection using this model self antigen that usually  
 induces tolerance, and whether Ad vector-directed production of  
 interleukin-2 (IL2) might augment this response. To evaluate this  
 approach, Ad vectors were constructed containing the murine gp75  
 cDNA (Ad.gp75) and the human IL2 cDNA (Ad.IL2). Efficacy was  
 evaluated in C57BI/6 mice challenged i.v. with 10(5) B16 cells, using the  
 number of lung metastases as the efficacy parameter. Naive control mice  
 developed 175 +/- 12 metastases by day 14. Controls receiving intranasal  
 Ad.IL2 1 day after B16 cell injection, intraperitoneal (i.p.)  
 mitomycin-C-treated B16 cells +/- i.p. Ad.IL2 before B16 cell challenge  
 and Ad.beta gal-treated mice had similar numbers of metastases as  
 controls  
 (P > 0.1). In marked contrast, preimmunization with intradermal Ad.  
 gp75 provided dramatic reduction in the number of lung metastases  
 (52 +/- 7, 29% of control). Addition of regional (intranasal delivery to  
 the lung) Ad.IL2 to intradermal Ad.gp75 preimmunization 1 day  
 following tumor challenge provided further protection (18 +/- 6, 10% of  
 control). Depletion of CD4+ and CD8+ T-cell subsets effectively blocked  
 the protective effect seen following immunization. Adoptive transfer of  
 macrophage-depleted splenocytes from Ad.gp75-immunized mice  
 similarly afforded significant protection against B16 tumor cell  
 challenge. Further, serum obtained 21 days following Ad.gp75  
 immunization showed no detectable anti-gp75 antibody by  
 immunoprecipitation. These results suggest that immunization with Ad.  
 gp75 induces cellular immune responses that

are capable of rejecting B16 melanoma in a host that is usually tolerant to **gp75 antigen**.

L33 ANSWER 3 OF 7 MEDLINE

DUPLICATE 2

1998118567 Document Number: 98118567. PubMed ID: 9435247. Fc receptors are required in passive and active immunity to melanoma. Clynes R; Takechi

Y; Moroi Y; Houghton A; Ravetch J V. (Laboratory of Molecular Genetics and

Immunology, Rockefeller University, New York, NY, USA.. clynesr@rockvax.rockefeller.edu) . PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1998 Jan 20) 95 (2) 652-6. Journal code: PV3; 7505876. ISSN: 0027-8424. Pub. country: United States. Language: English.

AB Effective tumor immunity requires recognition of tumor cells coupled with the activation of host effector responses. Fc receptor (FcR) gamma-/- mice, which lack the activating Fc gamma R types I and III, did not demonstrate protective tumor immunity in models of passive and active immunization against a relevant tumor **differentiation antigen**, the brown locus protein **gp75**. In wild-type mice, passive immunization with mAb against **gp75** or active immunization against **gp75** prevented the development of lung metastases. This protective response was completely abolished in FcR gamma-deficient mice. **Immune responses** were intact in gamma-/- mice because IgG titers against **gp75** develop normally in gamma-/- mice immunized with **gp75**. However, uncoupling of the Fc gamma R effector pathway from antibody recognition of tumor **antigens** resulted in a loss of protection against tumor challenge. These data demonstrate an unexpected and critical role for FcRs in mediating tumor cytotoxicity in vivo and suggest that enhancement of Fc gamma R-mediated antibody-dependent cellular cytotoxicity by inflammatory cells is a key step in the development of effective tumor immunotherapeutics.

L33 ANSWER 4 OF 7 BIOSIS COPYRIGHT 2001 BIOSIS

1997:331723 Document No.: PREV199799630926. **Immune response**

to melanosomal **differentiation antigen** induced by altered **antigen**. Bartido, Shirley M.; Hara, Isao; Naftzger, Clarissa; Wang, Sigun; Weber, Lawrence; Yang, George; Xu, Yiqing; Setaluri, Vijayasaraadhi; Qin, Jie; Moroi, Yoichi; Houghton, Alan N.. Memorial Sloan-Kettering Cancer Cent., New York, NY USA. Pigment Cell Research, (1997) Vol. 10, No. 1-2, pp. 103. Meeting Info.: XVIth International Pigment Cell Conference Anaheim, California, USA October 29-November 1, 1996 ISSN: 0893-5785. Language: English.

L33 ANSWER 5 OF 7 MEDLINE

DUPLICATE 3

97121471 Document Number: 97121471. PubMed ID: 8962137. **Immune response** to a **differentiation antigen** induced

by altered **antigen**: a study of tumor rejection and autoimmunity. Naftzger C; Takechi Y; Kohda H; Hara I; Vijayasaraadhi S; Houghton A N. (Swim Across America Laboratory, Memorial Sloan-Kettering Cancer Center, New York, NY 10021, USA. ) PROCEEDINGS OF THE NATIONAL ACADEMY OF

SCIENCES

OF THE UNITED STATES OF AMERICA, (1996 Dec 10) 93 (25) 14809-14. Journal code: PV3; 7505876. ISSN: 0027-8424. Pub. country: United States.

Language: English.

AB Recognition of self is emerging as a theme for the immune recognition of human cancer. One question is whether the immune system can actively respond to normal tissue autoantigens expressed by cancer cells. A second but related question is whether immune recognition of tissue autoantigens can actually induce tumor rejection. To address these issues, a mouse model was developed to investigate **immune responses** to a melanocyte **differentiation antigen**, tyrosinase-related protein 1 (or **gp75**), which is the product of the brown locus. In mice, immunization with purified syngeneic **gp75** or syngeneic cells expressing **gp75** failed to elicit antibody or cytotoxic T-cell responses to **gp75**, even when different immune adjuvants and cytokines were included. However, immunization with altered sources of **gp75 antigen**, in the form of either syngeneic **gp75** expressed in insect cells or human **gp75**, elicited autoantibodies to **gp75**. Immunized mice rejected metastatic melanomas and developed patchy depigmentation in their coats. These studies support a model of tolerance maintained to a melanocyte **differentiation antigen** where tolerance can be broken by presenting sources of altered **antigen** (e.g., homologous xenogeneic protein or protein expressed in insect cells). **Immune responses** induced with these sources of altered **antigen** reacted with various processed forms of native, syngeneic protein and could induce both tumor rejection and autoimmunity.

L33 ANSWER 6 OF 7 MEDLINE

DUPLICATE 4

96042160 Document Number: 96042160. PubMed ID: 7595233. Implicating a role for immune recognition of self in tumor rejection: passive immunization against the brown locus protein. Hara I; Takechi Y; Houghton A N. (Memorial Sloan-Kettering Cancer Center, New York 10021, USA. ) JOURNAL OF EXPERIMENTAL MEDICINE, (1995 Nov 1) 182 (5) 1609-14. Journal code: I2V; 2985109R. ISSN: 0022-1007. Pub. country: United States. Language: English.

AB The immune system can recognize **differentiation antigens** that are selectively expressed on malignant cells and their normal cell counterparts. However, it is uncertain whether immunity to **differentiation antigens** can effectively lead to tumor rejection. The mouse brown locus protein, **gp75** or tyrosinase-related protein 1, is a melanocyte **differentiation antigen** expressed by melanomas and normal melanocytes. The **gp75 antigen** is recognized by autoantibodies and autoreactive T cells in persons with melanoma. To model autoimmunity against a melanocyte **differentiation antigen**, mouse antibodies against **gp75** were passively transferred into tumor-bearing mice. Passive immunization with a mouse monoclonal antibody against **gp75** induced protection and rejection of both subcutaneous tumors and lung metastases in syngeneic C57BL/6 mice, including established tumors. Passive immunity produced coat color alterations but only in regenerating hairs. This system provides a model for autoimmune vitiligo and shows that **immune responses** to melanocyte **differentiation antigens** can influence mouse coat color. Immune recognition of a melanocyte **differentiation antigen** can reject tumors, providing a basis for targeting tissue autoantigens expressed on cancer.

L33 ANSWER 7 OF 7 CAPLUS COPYRIGHT 2001 ACS DUPLICATE 5  
1994:215142 Document No. 120:215142 Differential recognition of sequences within the encephalitogenic region of myelin basic protein capable of eliciting cell-mediated **immune responses** in experimental autoimmune encephalomyelitis. Malotky, Michele K.H.; Paterson, Philip Y.; Miller, Stephen D. (Med. Sch., Northwestern Univ., Chicago, IL, 60611, USA). J. Neuroimmunol., 48(2), 135-42 (English)

1993.  
CODEN: JNRIW. ISSN: 0165-5728.

AB The fine specificity of myelin basic protein (MBP) epitopes capable of eliciting in vivo delayed-type hypersensitivity responses in Lewis rats with exptl. autoimmune encephalomyelitis (EAE) was compared to those eliciting in vitro **antigen**-specific T cell proliferation and augmentation of disease transfer. Utilizing a panel of synthetic peptides

with sequences representing the 68-86 region of guinea pig (GP-) or bovine

myelin basic protein (B-MBP), animals were primed with one species of peptide and subsequently challenged with either the same peptide or peptides with truncations or substitutions representative of the other species of MBP. In regard to minimal length sequences capable of eliciting delayed-type hypersensitivity (DTH), rats primed with GP-MBP

and complete Freund's adjuvant (CFA) exhibited a hierarchical pattern of responsiveness to challenge with a series of truncated peptides, ranking as follows: GP-68-86 > GP-72-86 > GP-68-84 .mchgt. **GP-75** -86 = no activity. This response pattern corresponds to that previously reported for T cell proliferation and activation for disease transfer. A comparison of these T cell-mediated immune parameters, as elicited by the substituted peptides, revealed the response patterns of DTH reactivity to be similar to that previously described for in vitro T cell proliferation with significant DTH responses generated only by the peptide species for which the animal was primed. In contrast, a cross-reactive pattern of recognition was obsd. in cells mediating disease transfer, with all four 68-86 sequences capable of augmenting activation for adoptive transfer of disease, regardless of the peptide species for which the animal was primed. The **differential antigen** recognition patterns obsd. for these EAE-assocd. **immune responses** supports the hypothesis that multiple TH cell subsets are involved in disease pathogenesis.

'IN' IS NOT A VALID FIELD CODE  
L34 262 FILE MEDLINE  
L35 203 FILE CAPLUS  
L36 332 FILE BIOSIS  
'IN' IS NOT A VALID FIELD CODE  
L37 245 FILE EMBASE  
L38 34 FILE WPIDS  
L39 1 FILE JICST-EPLUS  
'IN' IS NOT A VALID FIELD CODE  
L40 434 FILE SCISEARCH

TOTAL FOR ALL FILES

L41 1511 HOUGHTON A?/AU, IN

'IN' IS NOT A VALID FIELD CODE  
L42 0 FILE MEDLINE  
L43 0 FILE CAPLUS  
L44 0 FILE BIOSIS  
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L45 0 FILE EMBASE  
L46 0 FILE WPIDS  
L47 0 FILE JICST-EPLUS  
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L48 0 FILE SCISEARCH

TOTAL FOR ALL FILES

L49 0 NAFTGER C?/AU, IN

'IN' IS NOT A VALID FIELD CODE  
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L51 0 FILE CAPLUS  
L52 0 FILE BIOSIS  
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L53 0 FILE EMBASE  
L54 0 FILE WPIDS  
L55 0 FILE JICST-EPLUS  
'IN' IS NOT A VALID FIELD CODE  
L56 0 FILE SCISEARCH

TOTAL FOR ALL FILES

L57 0 SATALURI V?/AU, IN

'IN' IS NOT A VALID FIELD CODE  
L58 143 FILE MEDLINE  
L59 61 FILE CAPLUS  
L60 99 FILE BIOSIS  
'IN' IS NOT A VALID FIELD CODE  
L61 85 FILE EMBASE  
L62 31 FILE WPIDS  
L63 2 FILE JICST-EPLUS  
'IN' IS NOT A VALID FIELD CODE  
L64 93 FILE SCISEARCH

TOTAL FOR ALL FILES

L65 514 GREGOR P?/AU, IN

=> s 165 and 141

L66 1 FILE MEDLINE  
L67 1 FILE CAPLUS  
L68 1 FILE BIOSIS  
L69 1 FILE EMBASE  
L70 0 FILE WPIDS  
L71 0 FILE JICST-EPLUS  
L72 2 FILE SCISEARCH



TOTAL FOR ALL FILES  
L73 6 L65 AND L41

=> dup rem 173  
PROCESSING COMPLETED FOR L73  
L74 2 DUP REM L73 (4 DUPLICATES REMOVED)

=> d cbib abs 1-2;s naftzger c?/au,in;s sataluri v?/au,in

L74 ANSWER 1 OF 2 SCISEARCH COPYRIGHT 2001 ISI (R)  
2001:75194 The Genuine Article (R) Number: 372WB. Measurement by Elispot of interferon-gamma secreting T cells before and after T cell depletion in donor bone marrow grafts.. Schaed S G (Reprint); Alpdogan O; Trcka J; **Gregor P D; Houghton A N**; O'Reilly R J; Collins N H. Mem Sloan Kettering Canc Ctr, New York, NY 10021 USA. BLOOD (16 NOV 2000)

Vol. 96, No. 11, Part 1, pp. 183A-183A. MA 786. Publisher: AMER SOC HEMATOLOGY.  
1900 M STREET. NW SUITE 200, WASHINGTON, DC 20036 USA. ISSN: 0006-4971.  
Pub. country: USA. Language: English.

L74 ANSWER 2 OF 2 MEDLINE DUPLICATE 1  
2000306559 Document Number: 20306559. PubMed ID: 10850386. Injection of DNA encoding granulocyte-macrophage colony-stimulating factor recruits dendritic cells for immune adjuvant effects. Bowne W B; Wolchok J D; Hawkins W G; Srinivasan R; **Gregor P**; Blachere N E; Moroi Y; Engelhorn M E; **Houghton A N**; Lewis J J. (Memorial Sloan-Kettering Cancer Center, New York, NY 10021, USA. ) CYTOKINES, CELLULAR AND MOLECULAR THERAPY, (1999 Dec) 5 (4) 217-25. Journal code: CUS; 9713367. ISSN: 1368-4736. Pub. country: ENGLAND: United Kingdom. Language: English.

AB An important issue for effective vaccines is the development of potent adjuvants that can facilitate induction or augmentation of immunity. Granulocyte-macrophage colony-stimulating factor (GM-CSF) is a growth factor for myeloid progenitors of monocytes and dendritic cells (DC), which upon maturation are antigen-presenting cells (APC). The adjuvant effects of inoculation of DNA encoding GM-CSF into skin were studied. Initial experiments examined whether the GM-CSF gene injected into the skin of mice could affect the density of epidermal DC (Langerhans cells). DNA encoding GM-CSF delivered by particle bombardment into skin resulted in a significant increase of epidermal DC at the inoculation site.

Kinetic analysis of epidermal recruitment after GM-CSF inoculation showed an increase in DC that peaked at seven days. This increase was accompanied

by recruitment of DC into draining lymph nodes. The adjuvant effects of DNA encoding GM-CSF inoculated into skin were measured by the ability to augment antibody and T-cell responses against poorly immunogenic tumor antigens. Peptide immunization at skin sites containing epidermal DC

newly recruited by GM-CSF DNA elicited T-cell responses against mutant p53, whereas peptide immunization of control skin sites did not elicit any detectable T-cell responses. Likewise, generation of antibodies following immunization with DNA encoding human gp75TRP1, a tyrosinase family member

expressed by melanomas, was accelerated and protection from tumor challenge augmented by GM-CSF DNA.

'IN' IS NOT A VALID FIELD CODE  
L75 5 FILE MEDLINE  
L76 5 FILE CAPLUS  
L77 9 FILE BIOSIS  
'IN' IS NOT A VALID FIELD CODE  
L78 5 FILE EMBASE  
L79 1 FILE WPIDS  
L80 0 FILE JICST-EPLUS  
'IN' IS NOT A VALID FIELD CODE  
L81 4 FILE SCISEARCH

TOTAL FOR ALL FILES  
L82 29 NAFTZGER C?/AU,IN

'IN' IS NOT A VALID FIELD CODE  
L83 0 FILE MEDLINE  
L84 0 FILE CAPLUS  
L85 0 FILE BIOSIS  
'IN' IS NOT A VALID FIELD CODE  
L86 0 FILE EMBASE  
L87 0 FILE WPIDS  
L88 0 FILE JICST-EPLUS  
'IN' IS NOT A VALID FIELD CODE  
L89 0 FILE SCISEARCH

TOTAL FOR ALL FILES  
L90 0 SATALURI V?/AU,IN

=> s 182 and 165 and 141  
L91 0 FILE MEDLINE  
L92 0 FILE CAPLUS  
L93 0 FILE BIOSIS  
L94 0 FILE EMBASE  
L95 0 FILE WPIDS  
L96 0 FILE JICST-EPLUS  
L97 0 FILE SCISEARCH

TOTAL FOR ALL FILES  
L98 0 L82 AND L65 AND L41

=> del his y

=> fil reg

COST IN U.S. DOLLARS

FULL ESTIMATED COST

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

SINCE FILE	TOTAL
ENTRY	SESSION
78.27	79.18

SINCE FILE	TOTAL
ENTRY	SESSION